current will dull the sensation which was awakened by a mild one. The sensation returns very rapidly and the temperature of the parts rises as does the muscular power. But chiefly in cases of aphasia, amaurosis and hemichorea, the actual cautery applied to the nape of the neck is exceedingly useful, the instrument to be heated to whiteness and not allowed to burn the skin—to be used with a light hand and often repeated. When there are traces of inflammatory processes of the membranes or of the nervous tissues, blisters of mustard on the points of the spinal cord tender to the touch are very useful, and ergot internally, or belladonna, etc.

These are the general indications, which, I trust, are in accordance with what I believe is the pathology of the diseases considered, and which have, besides, when used empirically also afforded good results.

37 W. 33D ST., NEW YORK CITY.

ART. III.—THE PSYCHOLOGICAL PATHOLOGY OF PROGRESSIVE PARESIS.

BY EDWARD C. SPITZKA, M.D.

(*Read before the Neurological Society of New York City, Feb.* 5, 1877.)

IN the choice of progressive paresis, for my subject, I was determined, not so much by the intrinsic interest of this affection, as by the fact that through its pronounced and constant characters it furnishes the best illustration of certain general propositions, whose enunciation is my chief object this evening. I do not intend to give a detailed and continuous account of either the clinical symptoms or the morbid lesions of progressive paresis, but rather to exhibit the physiological relation existing between these two factors.

As this is a comprehensive and controversial subject, it necessitates a reference to almost every attainment of modern nerve physiology; and I have therefore thought it well, in order to avoid infringing on the subject proper by the discussion of side issues, to briefly define my position with reference to some recent views, as a preliminary.

Among those diseases which may be considered pathological guages of the value of the experiments performed by Hitzig and Ferrier, the subject of this paper is certainly one. I myself began my investigations into the pathology of this disease, with an expectation of finding localization of the morbid processes, in particular areas of the cortex, corresponding to the motor peripheries chiefly involved. Naturally, these expectations were only partly realized; at the same time I should state that I was never convinced of the conclusiveness of Hitzig's experiments. There are too many sources of error in his methods, and that author has himself recently confessed that, on endeavoring to verify his conclusions by extirpating his so-called motor centres, he obtained negative results alone! I do not object to Hitzig's deductions on the same grounds which several French investigators have urged, namely, the supposed action of the electric current on the nerves of the pia mater! Even if this took place, the general truth of the principle of localization, would not be thereby invalidated, for these experiments could only gain in value if it should be shown that they acted by causing a local hyperæmia, strictly comparable to a functional determination of blood towards an active centre; an experiment which so closely imitated natural conditions would be the very ideal of the physiologist!

The exceptions to be taken are of a different nature, they are founded partly on the diffusibility of currents in a parenchyma, close to which conducting strands run in all possible directions, partly on the intimate connection, which we are forced to admit *a priori*, as existing between motor and sensory cortical areas.

Leaving the question, as to our ability to localize motor centres, in the lower animals experimentally, I strongly dispute whether, this being accomplished, we would be justified in transferring the results obtained to the human brain without further ceremony.

Attempts of this kind have been made on the strength of the homology in convolutional types among the Mammalia, which has been established by Huxley, Flower, Wernicke, Leuret, and other comparative anatomists. While this general morphological correspondence is unquestionable, it is not so decided, that anatomically homologous regions are necessarily physiologically homologous. If the development of the convolutions bears any constant relation to their motor function, we should expect that particular convolutions will rise and sink in their relative dimensions and complexity, with the variations in bulk and importance of the corresponding motor periphery. (In the kangaroo, for example, we would expect to find the centre for the hinder extremities to preponderate over the homologous area in the mole.)

This expectation is not fulfilled,* and with this, the transferribility of motor centres from one species of animals to another falls to the ground.

I could multiply examples, which all would show that the convolutional type does not depend so much on the habits and functions of a species, as on the powerful hereditary influence which has been transmitted to all descendants of a common stock, by an ancient ancestral race in bygone geological periods. The convolutional peculiarities depend on genetic and evolutional expressions of mechanical laws of growth, not on subsequent specific functional modifications.

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*There is a mammal, inhabiting the rivers of Africa and South America, which furnishes a test case; namely: the Manatus. This creature corresponds closely to the porpoise, both in its muscular periphery and general outline, for both animals are constructed with reference to an aquatic residence. On analyzing those structural affinities, however, which are alone conclusive in classification, we find that the manatus belongs to the pachydermata, and is consequently related to the elephant, than which a more dissimilar animal, as to shape and muscular contrivances, cannot well be imagined. Now on comparing the cerebral hemispheres of the manatus with those of the elephant on the one hand, and those of the porpoise on the other, we find that the resemblance is greatest in the former instance. In short, the convolutions are most similar in two animals, whose motor periphery is most unlike, in contradistinction to the dissimilarity existing between the convolutional distribution in two animals, whose motor periphery is the same. This bears out what is stated above.

We can easily afford to pass by the English writer's views without further discussion. Aside from the fact that he has merely adopted Hitzig's method and idea without appropriate acknowledgement, his only claim to originality is to be found in the fact that by increasing the strength of the currents employed, he has increased his sources of error. There is one startling exposition to be found in his first monograph. In the sixth experiment, on irritating a certain area of the second frontal gyrus, the dog experimented on, got up, looked fixedly in one direction, wagged his tail and fawned; Ferrier leaves the reader to form the conclusion that he has set an intellectual centre into action by characterizing these actions as an acted dream, and, in fact, he registers this mental centre with label No. 14.

He is evidently not aware that many animals perform the same and similar movements under chloroform, even when the brain is not touched, and every surgical operator is familiar with the co-ordinated actions, nay whole sentences spoken by human beings during the progress of an anæsthesia.

As to the more exact and reliable localization of functional areas, by anatomical methods, it is the only one which promises a satisfactory solution, of the difficult problem of normal and morbid cerebration. Its teachings should form the basis of every attempt, like the present one; to refer mental and motor symptoms to cerebral lesions.

Each area of the cerebral cortex is connected with the outer world, through medullated nerve fibres, which by their mediate connection with the peripheral nerves, terminate in the muscles and the peripheral "end-organs" of sense. It is consequently evident, that the fibres of the corona radiata represent both centripetal sensory and centrifugal motor tracts, and that, if it will ever be possible to trace the fasciculi of white substance from the nuclei of the nerves, to the cerebral cortex, we shall undoubtedly be able to map out the convolutions into functional centres, if these exist in the strict sense of the term. Such a continuity can not be traced directly, for ganglionic masses are interposed, interrupting the course of the fibres at various topographical altitudes. Our future exacter knowledge of cerebral physiology, depends on

the unravelling of this Gordian knot of associating commissural and decussating fasciculi. The first step in the right direction was taken by Luys and Stilling, and Meynert and Flechsig have more recently and more successfully undertaken the same task.

Meynert found that the great medullary tract through which each hemisphere is connected with the lower centres and the peripheral nerves, the crus, is naturally divisible into an upper tegmental and a lower basal portion, the pes pedunculi. Through each of these the prosencephalon is connected with the periphery, by the intervention of different ganglionic systems. The upper tract contains those fibres which have passed through or been interrupted in their course by the corpora quadrigemina and the thalamus opticus, he therefore terms these ganglia, tegmental ganglia, in contradistinction to the corpus striatum and nucleus lenticularis, which because they are connected with the basal tract, he distinguishes as ganglia of the pes pedunculi. It is through the ganglia of the tegmentum, that, besides other functions, the higher automatic actions are carried on; by the corpora quadrigemina, for instance, the regulation of locomotion, in reference to our visual impressions (through the lemniscus); by the thalamus, its regulation with regard to the tactile impressions. Through the ganglia of the pes pedunculi on the other hand, voluntary impulses are transmitted to the periphery. Sensory impressions are forwarded by both in a different degree.

If we proceed to trace the two medullary systems downward, through the pons, we find that the fibres of the pes pedunculi are directly continuous with the anterior pyramids. Now as you are aware, the anterior pyramids undergo a double decussation by the upper fibres, cross through the medulla to reach the opposite funiculus gracilis and funiculus cuneatus. These fibres are consequently continuous with the posterior columns of the cord, which proves the upper decussation to be of a sensory nature. By the lower, fibres are sent to the opposite lateral columns, while such fibres as do not cross at all, are continued downwards, as part of the anterior columns of the cord. (I must distinctly reiterate here, that while there are all possible variations in the decussation, this decussation is

never a complete one, as some recent compilers seem to believe, in ignorance of the researches of Lockhart Clarke, and Meynert). These fibres, together with those constituting the lower decussation are therefore motor.

On retracing these fasciculi to where they form an integral part of the pes pedunculi, we find that those which participate in the sensory decussation, are represented by the *outermost* fibres of the pes, the motor fibres by the *innermost* and middle portion.

Proceeding still higher to where the fibres of the pes pedunculi enter the ganglia and the internal capsule, we perceive that the outermost sensory ones run through the corona radiata to terminate in the occipital lobe, by a bold sweep backwards. I have been able to trace this fasciculus, which loses itself in a fan-shaped scattering of fibres to the posterior part of the gyrus fornicatus, the cuneus, and the praecuneus. From this fact there is but one conclusion to be drawn, namely, that the occipital lobe is subservient to the sense of touch, for no other sense can be projected through a tract which is continuous with the posterior columns of the spinal medulla. As Gratiolet had already shown, those ganglia into which the optic tract enters, which are mainly the corpora quadrigemina and geniculata, are connected with the occipital and posterior part of the parietal lobes by a powerful fasciculus, the "*radiations optiques.*" We will consequently locate the conscious centres of visual impressions in this area, also. Burdach, Gratiolet, and Meynert have further shown, that the white anterior commissure which is in reality an *olfactory chiasm*, is also connected with these cortical areas by a slender fasciculus. It is a natural conclusion then to attribute to the occipital lobe, a definite relation with the sense of smell. We thus see that the occipito-parietal lobe possesses three decided sensory connections. The frontal lobe with the island of Reil and operculum, is connected through the anterior part of the internal capsule, and the motor ganglia with the *innermost* area of the pes pedunculi; the old experimental and pathological concluclusion that these cortical areas are motor territories, is thus anatomically confirmed.

The lenticular nucleus receives its fibres from the island of

Reil and operculum chiefly, the fasciculus which leaves its lower border passes close to the median line, forming *the most internal fibres of the voluntary motor tract.* Meynert concludes from this that these fibres form those nearest the median line in order to be the first to cross the raphe and place the motor nuclei of the cranial nerves under the crossed influence of the opposite hemisphere. His surmise is, that as this bundle represents the motor cranial nerves, the cortical area, above mentioned, from which it originates, is the centre for the nerves of expression, both phonetic and symbolic.

But while it is quite clear that the frontal lobe is eminently motor in its connections, we cannot yet assert that it is exclusively so; as little can it be said that the occipito-parietal lobe is solely sensory. The tegmental ganglia on the one hand receive fibres even from the most anterior part of the frontal lobes, while the caudex of the corpus striatum is in a connection, however slight, with the extreme occipital end. I should consequently be inclined to characterize the functional topography of the convolutions, not so much as a sharp, abrupt demarcation of various centres, but rather as a blending, a dovetailing, in which there are marked foci of functional concentration.

On proceeding to examine more intimately, the distribution of the fibres to the cortex, we find that two kinds of fibres terminate in the latter. Those which formed a part of the corona radiata enter the gray substance in bundles, which spread out fountain-like, the individual axis cylinders can frequently be traced to be directly continuous with the basal processes of the ganglionic cells. The other class consists of fasciculi, which connect the apex of each convolution, with the apex of its neighbor, by arching around the lowermost layer of cortical substance at the base of the sulci. It is an inference, in complete accordance with physical laws, to suppose these as well as similar fasciculi which join more distant areas to be associating tracts. That, furthermore, such associating tracts must play an important part in the mechanism of coherent thought and associated voluntary action need not be demonstrated.

It would lead me too far, to-night, to recount the philosoph-

ical explanation of thought and action, on a purely physical basis, which Meynert and Wundt have given, and which is quietly but surely gaining adherents on the continent, and which will, no doubt, revolutionize the thus far unsatisfactory and unscientific methods of explaining the phenomena of insanity. Suffice it to say, that our highest intellectual processes, according to these philosophers, depend on molecular changes in the nerve cell and axis cylinder, which are comparable to electro-negative oscillations; and that the more numerous and powerful the impressions, and motor innervations stored in the six hundred million of protoplasmic organisms, which people our cortex, and the more extensive their protoplasmic connections and the arched associating fasciculi, the more perfect will be that voluntary control over our impulses, which distinguishes the cultured man from the savage, the sane man from the lunatic!

And here we are approaching our subject. How are we to picture to ourselves the manner in which a morbid process causes aberrations from the normal standard of mental action? More particularly how does it produce the mental and motor phenomena, characteristic of progressive paresis?

The principles which should guide us in such an inquiry can be laid down in the following propositions:

I. Every manifestation of typical progressive paresis may be formulated as consisting in an altered activity of some one of the higher functions of the nervous system; the sole seat of these functions is in the prosencephalon, and it is obvious that in searching for the somatic basis of this alienation, our attention should be primarily directed to the region specified.

II. Whenever an anatomical or chemical change is discovered in the higher centres of an individual, dying with paresis, which can be shown to be independent of the manipulations by the histologist, as well as of the results of post mortem decomposition, we are justified in assuming such change to be the proximal cause of the symptoms manifested during life, on the part of the mind. Such an assumption becomes almost a demonstrated certainty, when a number of individuals, affected with the same or similar group of symptoms, exhibit the same or similar cerebral changes.

III. It is equally evident, since the prosencephalon is not a single organ, but an agglomeration of numerous, variously associated, and functionally, more or less differentiated centres, that it will be well to determine not alone the histological character of the morbid process, but also the precise topography of the region involved.

IV. Inasmuch as other portions of the cerebro-spinal axis, beside those concerned in conscious action and sensation, are occasionally involved in subjects of progressive paresis, and the resulting symptoms serve to mask or modify the clinical aspect of the disease, the automatic and reflex tracts extending from the third ventricle down to the filum terminale, should be carefully examined in order that their disorders may be differentiated from those of higher centres.

The first proposition is verified by the testimony of every competent observer. As to the second, it remains to be seen, since the essential lesion is undoubtedly cortical, whether the pathological change in progressive paresis is constant, and in what it differs from the pathological changes of other forms of insanity, and other hemispheric affections.

Our answer to this question depends on, what is all included under the term progressive paresis. Recent investigation has shown that the combination of mental and motor symptoms comprised under this head, does not present a uniform type, but that there are several varieties of the disease, independent of the fact that progressive paresis is occasionally a complication of chronic melancholia and chronic mania. If all these various forms be promiscuously thrown together, then I must confess that there is no particular morbid process characteristic of the disease. But if paralytic insanity be considered a generic type, under which several distinct species are admitted to exist, then I am fully prepared to maintain a unity of the pathological character for each such species.

(*a*) The first variety constitutes the classical picture of paresis, in which the mental symptoms appear first, and are soon followed by disturbances of the delicate muscular contrivances of the eyeball, face, tongue and pharynx, the extremities being involved last. Topographically speaking, this is a descending affection.

(*b*) The second variety is founded on numerous cases observed by Westphal, to which I can contribute several instances, in which the first symptoms are spinal, and the muscular co-ordinations presided over by the cranial nerves, are destroyed in the order in which their nuclei lie in the floor of the fourth ventricle and the aqueduct of Sylvius; the intellectual symptoms are the last to manifest themselves. This, in contradistinction to the former, is an ascending affection. I am inclined to consider the mental symptoms in this case as independent of the original affection, and altogether regard these cases as instances of locomotor ataxia, complicated by progressive paresis, although my investigations on this point are not yet concluded.

(*c*) We also see, though less frequently, a third group of cases in which the cerebral and spinal symptoms nearly coincide as to their time of appearance, the former more in the way of a progressive dementia, than of delusions of grandeur, the latter presenting itself as a progressive paralysis and ataxia of individual muscular groups. If it were clinically possible to separate this form from the others, it would be, pathologically speaking, desirable, for the lesion is a multiple cerebrospinal sclerosis, although the symptoms supposed to be characteristic of this process are ill-marked.

(*d*) Corresponding to the third form, as regards the synchronicity of the encephalic and the spinal symptoms, but materially different from it in that the mental aspect of the case is so like that of the typical affection as to be scarcely distinguishable from it, is one of the manifestations of the syphilitic dyscrasia. There are here, primary changes in the vessels, a subsequent infection of the adventitia, and, finally, destructive changes in the nervous parenchyma proper.

While these four varieties will have claimed the attention of every physician who has seen a large number of cases, there are others which are rare, if not unique. I include here more particularly a case, which exhibited all the symptoms of progressive paresis, but in the autopsy, presented multiple syphilomas scattered through the encephalon. As such a case, which first staggers one, by its exceptional character, furnishes an admirable argument in favor of the dictum, that symptoms

referable to the cerebro-spinal centres are less attributable to the microscopic character of a lesion, than to its topography and extent, I subjoin the necropsy, preceded by a brief history, for which latter, I am indebted to Drs. Kiernan and De Hart.

"The patient, a French laborer, was admitted to the City Asylum for insane males, at the age of twenty-six; he was luetic and intemperate.

"On reception, he exhibited delusions of grandeur of a rather stupid character; he maintained that he possessed vast amounts of movable and landed property, also that he was about to grow immensely in stature. At this time, he began to manifest a slight difficulty of articulation, and his tongue was tremulous. For the first two weeks, he was very excitable, so as to require large doses of sedatives. After this period, he became progressively more incoherent and irrelevant in conversation; he shouted at the top of his voice, without being moved thereto by any apparent cause, and refusing to take food for two days, as he imagined it to contain poison, was transferred to the hospital ward. The motor paresis had meantime increased to almost complete paraplegia of the lower extremities, while the labial tremor was extreme. He still retained his old delusions, more stupidly expressed than previously, to which he added that he was able to lift the building on his little finger, but he could hardly enunciate one continuous sentence uninterruptedly. A well-marked convulsive attack was noticed, and the difficulty of articulation having increased to well-nigh complete aphasia, death took place from exhaustion, following a maniacal attack.

"The most marked mental symptom manifested by this patient was a complete loss of certain recollections, a greater part of his existence while in France had become a complete blank to him, he had forgotten altogether that he had been a laborer. My attention was called to this case by Dr. Kiernan, more on account of a peculiar motor symptom than for any other reason, this symptom being the only one in which it varied from the ordinary course of progressive paresis. It was a passive contracture of the muscles of the neck and left arm, the chin being drawn to the left and backwards so as to

touch the left clavicle, the arm being partly flexed and pronated, and carried behind the back. On rousing him, he could abandon this constrained position, but gradually relapsed into it afterwards. From this I made the diagnosis of a left thalamus affection, locating the lesion in the posterior part of that ganglion in accordance with Meynert's diagnosis of the same symptom in an epileptic imbecile, in whose case the autopsy confirmed the diagnosis.

"In this case, I made the autopsy twelve hours after death.

"The cerebral dura was adherent to the cranium, and presented a greenish discoloration over the left lobulus tuberis, otherwise it showed nothing abnormal. The dura mater spinalis, however, was extremely thickened from the exit of the first to that of the fourth cervical pair, exhibiting other appearances of pachymeningitis. The leptomeninges of the encephalon were thickened and infiltrated with young cells, but no pus, coagulable lymph, or increase of the arachnoidal fluid could be determined. A similar, more intense process had affected the spinal membranes, and a diffuse, gummy infiltration, cheesy in its centre, involved the parts around the anterior fissure of the spinal medulla, opposite the roots of the third cervical pair, and had destroyed the anterior commissure of that district.

"The cortex of the hemispheres was everywhere the seat of various superficial and nodular infiltrations. The nodules were of all dimensions, from microscopic to half an inch or more in dimensions; the smaller were spherical and peri-adventitial, with respect to some vascular trunk; the larger were ovoid, their long axis represented by the stem of a large vessel, being vertical to the cortical superficies. The diffuse infiltration involved the uppermost layer of the cortex, and was chiefly located at the floor of the sulci, and especially over the island of Reil, as well as the corresponding inner surfaces of the operculum and temporal lobes.

"Where the nodular growths had become larger, they fused with the superficial infiltration, giving to the latter an appearance as if it had processes dipping into the deeper layers. Frequently several nodules situated on different branches of the same main vessel, fused with each other around the latter,

thus resembling a papilloma. The white substance was also affected, the nodules not being, however, so well marked, and the transition from the neoplasm to the normal tissue was gradual. Their color varied, as did also their consistency; while the smallest were undistinguishable by the naked eye from the surrounding tissue, medium sized nodules exhibited a greyish-reddish, or yellowish-white centre, with a markedly red zone at the periphery, these were quite firm. Larger ones showed the same peripheral zone, with a softening of the centre, and the last stage of the breaking-down process, which terminated the existence of these masses was represented by cavities of varying dimensions, some without any sharp boundary and softened walls, others provided with a more or less firm connective tissue capsule. The distribution of these masses was symmetrical in both hemispheres, but the softening process had proceeded further on the left than on the right side. The right thalamus was entirely free, while the left which was intact in its anterior half, was converted into one mass of anastomosing nodular and tubular infiltrations in its posterior tubercle. The focus of the change was in the lenticular nucleus and the island of Reil, as you can see in the specimen, which I hand around, by the naked eye. It represents a section through the territories mentioned, made by a microtome; the largest cavity, or rather gap, which you see, involves the claustrum, external capsule and the outer articulus of the nucleus lenticularis, the latter is actually riddled with cysts, the internal capsule is relatively free. Other regions which were affected in a high degree were the central extremities of both praecentral gyri; the lobulus tuberis and the cornu ammonis. The left tegmental tract was completely broken down; one large cyst occupied the place of the left olivary body, two smaller ones were situated in its fellow. There were besides several small, diffuse, and for the most part softening nodules in the cerebellar hemispheres likewise quite symmetrical.

"The microscopic examination showed the youngest tumors to consist of an accumulation of round mononucleated cells, varying in size from the dimensions of a red corpuscle to those of a white one. These were situated in the adventitial sheaths of the vessels, and gradually encroaching on the perivascular

space, obliterated the latter and penetrated into the neighboring neuroglia. Now the neoplasm began to assume distinctive characters; the main body was composed of older elements which did not imbibe carmine well, and stiff, coarse fibres, pyramidal nerve cells, with intact contours, were still to be seen in the very midst of the mass; the peripheral zone was composed of young elements of the nature described above, as well as free nuclei, which were rapidly and deeply stained by carmine. Later on, the centre of the nodule underwent a partly mucoid, partly granular disintegration; in the resulting detritus mutilated nerve cells were yet visible. The neoplasm, whose periphery exhibited a rich vascularity, was determined to be a miliary syphiloma.

"It is unquestionable, that if the patient had not died from exhaustion, we should have attributed his death to the process which destroyed the important centres of the medulla and tegmentum.

"We here had a neoplasm, by which its multilocular occurrence produced those symptoms usually supposed to be associated with the chronic inflammatory process characteristic of typical progressive paresis. As we shall show further on, the elements in this inflammation which are productive of the morbid symptoms, are due, on the one hand, to anomalies of the circulation, and on the other to localized destructive processes. Notwithstanding its widely different histological character, this neoplasm acted in the same way; by its presence it kept up an irritative hyperæmia, and through its proper necrotic processes caused localized destructions in the cortex, the ganglia, and medullary tracts.

"As to that symptom, noticed as a permanent change in the patient's position, and which had led me to diagnosticate a thalamus affection during his life, it could not be so clearly referred to that ganglion after the thorough post mortem examination had been made. It is true the left thalamus was diseased, and that portion which had been surmised to be affected. But there was also a lesion of the left tegmentum, as well as of the spinal cord; there are thus three lesions to choose from, either one of which might have produced the symptom in question.

"The aphasia, in like manner, could not, as far as its ataxic element was concerned, be referred to the island of Reil or operculum, for the left hypoglossal nerve was totally destroyed by the large area of softening in the left olive."

In subjects of typical progressive paresis who die through intercurrent affections in the first stage of the disease, the only pathological change I could discover was a certain degree of leptomeningitis, and a dilatation of the perivascular lymph-spaces in the cortex. Neither of these changes is in any way pathognomonic of progressive paresis, for meningitis is found indifferently in other forms of alienation, as well as in sane persons, while dilatation of the perivascular spaces is much better marked in chronic alcoholism, melancholia, and epilepsy of long standing. These two changes have an etiological bearing, however, especially the latter, which, by weakening the lateral support of the vascular walls, throws an increased strain on the centres which regulate the vascular tonus, so that these breaking down, cause the hyperæmias which we shall refer to, and by recruiting their powers, determine the return of the vascular current to approximately normal conditions, and with this an amelioration for the time being of the symptoms.

Where, however, the patient has died in consequence of exhaustion from a maniacal outbreak, or has died from other causes shortly after such a one, we find a high degree of engorgement in the cerebral capillaries; at the same time proteinaceous bodies are found in the neuroglia, which, it seems, can be rapidly removed by the lymph channels. These bodies are identical with the so-called colloid bodies of authors, and are to be regarded as a true exudation. It is true the nuclei of the neuroglia are frequently concerned in their formation, but this occurs secondarily to the production of the material in an amorphous condition, the neuroglia cell takes it up as a morbid pabulum, and thus the round bodies familiar to cerebral pathologists seem to be produced. In many early fatal cases, such as die in the first and second stages of the malady, and which, on account of the violent motor excitement, rapid flight of ideas, and the fact that the paresis is not well developed, are frequently confounded with acute mania, in some of our asylums, these bodies are found even down in the commencement of the spinal medulla. Smaller ones join to form

larger accumulations, forming opaque spots visible even to the naked eye. Such changes, on account of their obvious fatality, have no psychological interest for us.

I do not hesitate to attribute the rapid flight of ideas to this hyperæmia, further its incoherent character to the fact that in certain cortical areas the engorgement reaches a higher degree than in others, producing an actual stasis, which suffices to render certain recollections, forming part of the patient's "ego," by which he appreciates his proper relations to his past and present, nugatory. I have frequently found partial stasis in the cortex of patients who had died in a stupid condition, combined with excitability. As the disease progresses, it seems that the vessels, badly supported in the dilated perivascular spaces, and subjected to repeated overstrain by repeated engorgements, suffer in resisting power, so as to become unable to discharge themselves of their contents, and forward the accumulating blood-column; when the next hyperæmia occurs, we will thus have a genuine stasis, and this stasis is of a peculiar character; we find the blood corpuscles no longer distinguishable, but replaced by, or rather fused into, an opalescent hyaline cylinder. When resolution takes place, this cylinder breaks up into spherical or oval fragments, which are carried onwards in the vascular current, becoming further subdivided at each bifurcation, and finally are represented by a granular material having the same optical appearance as the larger masses. Meynert, who first describes this condition, brings it into relation with the stupor, and observes that, as the stasis follows the hyperæmia, so the stupor follows the excitement. This explanation is exceedingly plausible, and we may carry it still further, and say that the resolution of the stasis corresponds to the so-called lucid interval following the stupor; the possibility of a well-nigh perfect* re-establishment of the patient's normal mental condition, is readily comprehensible, when we regard the pathological changes as mainly nutritive, not destructive, in their character.

*I do not intend to convey the idea that the lucid interval is ever complete, and consequently give my fullest assent to Westphal's exceptions to Lubimoff's statement, but I must insist that the return to the approximately normal state, may be so nearly complete as to render the mental defect undiscoverable, save to the experienced physician.

There is no region of the brain which is so vulnerable to vascular determinations as the lenticular nucleus, for here the vessels are less supported than in any other portion of the encephalon. We consequently find that dilated and contorted vessels are nowhere so frequent as here, and it is a *locus prædelictionis* for the stasis described. Accordingly no symptom is so constant in progressive paresis as a disturbance of those delicate co-ordinations which are transferred through this ganglion, namely, the articulation and expression.

With the repetition of this vascular stasis, destructive and irreparable change takes place. This occurs both in those cases in whom frequent exacerbations and remissions point to rapid changes in the blood-supply, and in those where the more continuous course of the symptoms would lead us to picture to ourselves a more permanent, slowly-increasing and slowly-produced condition of engorgement. It is these permanent destructive changes which determine the peculiarly progressive character of the malady. With each engorgement clinically marked by an excited stage, the encephalon becomes more or less crippled. While in the earliest attacks the patient speaks with a rapidity which a sane man can hardly equal, and there is a truly wonderful flight of ideas, marked by a decided degree of energy, and even of creative fancy, we find that in subsequent attacks his flight of ideas and speech both become slower, and the patient is able to follow his ideas in writing, and finally contents himself with penning endless quires of disconnected and melancholy evidences of his dementia.

Meschede, in this connection, reports a case of a paretic who, when first brought into the asylum, suffered from a maniacal attack of thirty-six hours duration, in which the flight of ideas and rapidity of speech, amounted almost to a delirium. He did not interrupt the torrent of sentences which issued from him but once or twice, to moisten his parched lips with a drink of water, and all this time was engaged in measuring the orbs of the planets, the distance of the dog-star, squaring the circle, and he finally gave a feast to the whole world of truly Arabian Night's profusion. It was in a case of this kind, which died from exhaustion after such an attack, that I found the engorgement and colloid filtration alluded to.

The final destructive changes are of two kinds, those which affect the nerve elements directly, and those of whose damaging effect on the latter, we have no direct optical evidence but can judge inferentially.

The characteristic of these processes in progressive paresis is, that they are rarely general, but that they affect certain provinces of the cortex, and leave others entirely or nearly intact, as far as we are able to determine. Not only this, but there is the greatest difference in the affected localities. While some are so utterly disorganized as to leave only one inference open, that their function was completely cut out of the mental life of the individual, others present such slighter changes, as would lead us to infer that the corresponding functions were merely weakened. We should thus have a second parallel to establish between lesions and symptoms; the totally destructive process would correspond to complete blanks in the patient's memory, and the entire loss of control over definite muscular groups; the partially destructive ones to a dimness of recollection, and diminished control over the same motor periphery.

It is a significant fact, that those movements which depend on the voluntary association of smaller muscular territories, suffer before those which depend on the combination of coarser contrivances. It would seem to point to the fact that the smaller muscular peripheries were represented by smaller hemispheric areas than larger ones, and that lesions of slight extent could consequently involve the lesser more entirely, and with proportionately greater functional damage, than the larger ones. This is partly also to be referred to another fact, that the layer of the cerebral cortex, adjacent to the associating fasciculi, is the first to suffer.

Before proceeding to analyze the intimate changes of the cortex and their probable relation to the mental symptoms, I would summarize the extent of my observations on the localization of certain motor disturbances.

In patients whose disturbance of articulation was extreme, and had been the preponderating element during life, I found the changes concentrated about the island of Reil, the operculum, Sylvian aspect of the temporal lobe, and the lenticular

nucleus. In this extensive area, I have not been able to localize more narrowly. I drew this conclusion from three cases in which the medulla and peduncular tracts were intact.

In four cases (one, however, not an example of the typical affection), the paresis of the extremities, more particularly of the lower, was most marked. Here a territory corresponding to the upper extremities of the præcentral and postcentral gyri, as well as the contiguous convolutions, exhibited the most decided destructive lesions, with striking uniformity. I do not on the strength of a few observations form the conclusion that the centre for the extremities is here located, without some reservation, but simply record this, to me, striking relation, in order that it may be compared or contrasted with the observations of others; it is suggestive, however, when taken in connection with the fact that the innermost fasciculus of the middle portion of the internal capsule is connected with the short gyrus connecting the two border convolutions of Rolando's fissure, and the observation of Frey, and others, that softening and other lesions, in the track of this bundle of fibres, are associated with paralysis of the opposite arm or leg, or both. It is also this fasciculus which is the first to have its axis cylinders provided with a medullary sheath in the embryo, (Flechsig). We believe that those tracts develope first which are related to the first active periphery, and that the movements of the extremities are the first performed by the fœtus *in utero* to which a volitional element can be attributed, may not be insignificant in this connection.

Two cases which manifested no marked motor paresis, but well marked losses of certain recollections, exhibited destructive lesions of the second and third frontal gyri (first and second of Ecker), in one of these, however, there were also considerably affected areas in the occipital region. A third interesting example was afforded by one patient, who, from having been surgeon-dentist to Queen Victoria, was dismissed the court because of some ill-timed interference in the Lady Flora Hastings court scandal, became a general under Cavaignac in 1848, thence went to Australia, to become incarcerated for participating in a riot, and, coming to New York, was once or twice a candidate for the coroner's office, actually re-

ceiving votes. While he recollected many details of his strange career, both of his early life, and the most recent events, he had forgotten completely that he had been dismissed from the English court. He was a disciple of Mr. Henry Bergh, and declaimed violently against the cruelty of Magendie's experiments, showing a good recollection of their details which he had studied when a student at the University of Edinburgh. His brain exhibited lesions visible to the naked eye, in the third frontal gyrus and the adjoining part of the gyrus fornicatus. Pathologically, this case was not a pure one, for the whole peduncular tracts and spinal cord were the seat of a change, which corresponds partly to Lockhart Clarke's granular disintegration, partly to Arndt's observation of extreme luetic affection of the cerebro-spinal vessels. The whole tract of the hypoglossal and facial nerves was the seat of this disorganization, and his disturbance of articulation was referred to this lesion, not to the hemispheric affection, for neither his stock of words nor the construction of his sentences had suffered. It was merely the mechanical element which was interfered with.

As to the closing scene of progressive paresis—if the patient lives long enough—the whole convexity of the hemispheres becomes affected, mainly by a connective tissue hyperplasia, and subsequent atrophy of the convolutions. This corresponds to the general and complete character of the terminal dementia. It should not be forgotten in this connection, that some cases present from the very beginning, not special symptoms of obliviousness, or of motor paresis, but rather a general dementia and general paresis, its progress being one of degree. This is often the case with those whose symptoms are first spinal, as for instance where progressive paresis follows locomotor ataxia, and in some examples of the luetic form. Here we find a general affection of the convexity from the very beginning, when such cases reach the autopsy table through intercurrent affections in early stages.

It will require accumulated observations of this kind to determine the localization of marked and special symptoms, in this and other forms of cerebral disease, and I have offered the few deductions of localization of lesions in reference to dis-

tinct symptoms, not as so many unimpeachable facts, but because I think it better to hazard one rational conjecture, than to chronicle a score of disconnected details.

I should add that in progressive paresis, the lesions are strikingly symmetrical, with reference to both hemispheres, a fact in complete accordance with their anatomical and functional symmetry, and perhaps also with the observation of Arnold and Oellacher, that the corpus callosum connects corresponding convolutions of the opposite halves of the cerebrum.

There is a second group of symptoms, which without being either mental or volitionally motor, may be referred to the hemispheres. I refer to the so-called apoplectiform and epileptiform attacks, as well as to peripheral lesions.

Apoplectiform attacks, occurring in those cases, accompanied by that frequent complication, pachymeningitis, are most frequently due to a meningeal hemorrhage, and are in no manner different from other meningeal apoplexies. In a second class, particularly older patients, or such as are luetic, true parenchymatous hemorrhage into the oval centre of Vieussens, or the ganglia are found; occasionally softening is the lesion noticed. But a third category presents neither meningeal nor cerebral hemorrhage, nor softening, but merely an extreme condition of the stasis, previously described; in some instances this reaches almost the degree of a capillary apoplexy. Such a condition suffices to produce all the symptoms of an apoplectic attack, and I believe that it frequently precedes the true cerebral hemorrhages, acting as their exciting cause, by overstraining the weakened vascular walls.

In the last case, where I made an autopsy, a patient who two weeks before death became rapidly, but gradually paralytic on both sides, as well as comatose, shortly before death was suddenly seized with a deeper coma, his face purple, and breathing stertorous. I found a combination of hemorrhagic and necrotic conditions, which is rather unique. An extensive bloody suffusion of the whole pia mater was the chief morbid appearance of the convexity; at the base, an old organized extravasation, which had partly penetrated between the pia and the medulla, partly into the arachnoid space, in which it extended as far forward as the olfactory lobes. An enor-

mous extravasation was found in the left hemisphere, extending from between the thalamus and corpus striatum, upward and to both extremities of the hemisphere, so as to leave but a thin shell of cerebral substance, on the parietal and frontal lobes. The blood was mostly fluid, and the few clots present were soft and maroon colored; it was evidently a recent extravasation, and had distended the basal ganglia in the left lateral ventricle so as to obliterate the furrow which normally separates them. In addition three considerable depôts of softening were found, one in the apex of the left temporal, and one in each occipital lobe. Microscopical examination showed, besides the usual degenerative appearances, stasis, undergoing resolution, of all the cerebral capillaries.

On the whole, we may say, that many of the apoplectiform attacks, do not differ from the true apoplexies occurring in the same, and that they take place more frequently, because vascular changes and frequent vascular overstrain, the predisposing and exciting causes of cerebral hemorrhage, more often co-exist in progressive paresis, than among the other classes. Of the relation of vascular stasis pure and simple to pseudo-apoplectic attacks, frequently occurring in rapid succession, we have already spoken.

Before proceeding to the analysis of the epileptiform attacks, I would insist that these have nothing in common with the convulsive attacks of true epilepsy, except as regards the clonic convulsions, and even these do not correspond to the outbursts of typical epilepsy. In their reaction to nitrite of amyl, the two exhibit a fundamental difference. Crichton Browne first found that paretics are less susceptible than normal subjects to the influence of this "vascular neurotic," while epileptics are exceedingly rapidly affected by much smaller doses than will influence a healthy person. Correspondingly we find that if given under proper conditions, nitrite of amyl will abort a threatened epileptic onset, more surely than any other drug, while over the convulsions it exerts no good influence, on the contrary I should suspect it of having a bad one. This is to be explained by the opposite vascular condition obtaining in the two, in epilepsy the attack begins with a cerebral anæmia,

which is counteracted by the amyl nitrite, which causes a decided hyperæmia; in the parctic's convulsive attack we have a hyperæmia to start with and this, nitrite of amyl could only increase. If any medicine could be tried with an expectation of some success here, I should judge that drug to be ergotine. Meynert and Wedl have recently found the same arterial stasis affecting the cornu ammonis of epileptics which is found over the whole cortex of paretics, as well as in the cornu ammonis, and leads to degenerative changes in this territory. It is curious that the stasis of the cornu ammonis, should be arterial in epilepsy, especially when we find the remainder of the encephalon in an intense condition of venous congestion after the epileptic coma, and I think the clew to the nature of these attacks of true epilepsy and progressive paresis is here given.

In the *haut mal* we have a general cerebral anæmia resulting from the tonic spasm of the cerebral arteries, due to a hyperæsthesia of the vascular centres. Now there is one part of the cerebral cortex which is very favorably situated and constructed as it were for a collateral hyperæmia, this is just the cornu ammonis, which unlike any other cortical territory lies at the floor of the lateral ventricle, and possesses an anastomotic network of vessels in the stratum lacunosum. In contradistinction to the functional abolition of the remainder of the cortex, we here have a functional stimulus set up, which travelling down the fornix to the thalamus, determines the partly co-ordinated character of the convulsions. That the cornu ammonis is a motor territory I am satisfied on grounds of comparative anatomy and experiment. I will only mention here in passing, that this S-shaped involution of the cortex is proportionately better developed in the bat, an animal whose natural motions have the rapidity of epileptic convulsions, and in smaller Rodentia than in other classes.

In progressive paresis, this same hyperæmia in a degenerating territory, produces much feebler convulsions, while it is not impossible that in other cortical territories it may cause convulsions of individual muscular groups. That we have no total loss of consciousness as in epilepsy, has been recorded by every good observer, and I can, from a less extended experience, only confirm their statement. The condition of con-

sciousness, or even of excitability, which precedes the convulsion, passes uninterruptedly and progressively into stupor, this increasing with the progress of the convulsive attack, and extending beyond it; while in the epileptic attack we have: first, complete consciousness and aura; second, complete unconsciousness with convulsions; thirdly, complete coma, with occasional re-awakenings. With a radical difference between the vascular conditions in the two affections, the one being a vaso-motor spasm, the other a vaso-motor paralysis, we have an agreement as to the locality affected, in both it is as we have said the cornu ammonis. My observations in this respect are quite in accord with those of Meynert, and I can add the further observation that, in three cases presenting no convulsive attacks at all, the cornu ammonis was decidedly healthy, as compared with the same region in paretics who had convulsions.

Of peripheral organs affected, one of the most interesting is the retina.

The optic papilla is most constantly affected in that form of paralytic insanity, which complicates locomotor ataxia; in a case of this kind remarkable for the youth of the patient, who was only twenty-three, I found beginning atrophy of both discs. In an old standing case of genuine paresis, whose luetic nature could not, however, be excluded with certainty, there was sclerosis of the temporal side of the optic papilla, and a corresponding defect of vision in the right eye, while the left appeared healthy. A third case of advanced paresis, the distant result of an injury received at the battle of Bull Run, exhibited no retinal change whatsoever, while a fourth who began as a case of melancholia, with delusions of dread and persecution, and now presents decided paresis, general hyperalgia, tinnitus aurium and photopsia, had a markedly hyperæmic optic papilla. These four are selected cases, chosen to exhibit any possible relation between the retinal lesion and the symptoms; as could be anticipated, no such relation could be found, and where, as in the last case, delusions were founded on the photopsia, which, in its turn, was the result of a retinal hyperæmia—this hyperæmia had no more essential a relation to the delusions than circumstances originating outside of the patient's

body have, which equally serve to tinge his morbid ideas. To those who are accustomed to think physiologically it would be unnecessary to insist on this, I dwell on this point, rather because I have been informed that a few years ago an ophthalmologist went to one of our insane asylums to look for hallucinations and delusions in the retina! Which side of the mirror the hallucinations were on, in this case, I need not say.

My examinations of the optic nerve itself have been few, and consequently are not conclusive; but on looking over my records of autopsies, I am struck by the frequent observation of unusually firm and small optic tracts, chiasm and nerve. In one case I found numerous corpora amylacea scattered through these, and in a second, a decided and extreme hyperplasia of the perineurium and neuroglia, with atrophy of the axis cylinders and sheaths. I would not always refer the retinal affection to a descending neuritis or secondary atrophy, but hold it possible that the similarity of the retinal to the cerebral elements predisposes them to the same changes, the same engorgements and sclerosis. Another element in their causation may be the increased intracranial pressure, marked by the increase of the arachnoid fluid, and the chronic meningitis found in ninety-nine paretics out of a hundred. Perhaps the frequent co-existence of the luetic dyscrasia is not without some influence here.

One reason why the retina of paretics will never receive the attention it deserves, is that the fatality of the original affection renders it a matter of slight importance to diagnosticate an incurable affection. I will merely state that I have never met with complete amblyopia in a paretic, and that I consider Clifford Albutt's assertion, that where graver changes are absent, at least a hyperæmic blush of the papilla exists, as inaccurate.

Much more important, in a prognostic direction, are trophic disturbances of various distant organs. There is a great frequency of gangrene of the lungs, and of fragility of the bones, particularly the ribs, among paretics. These, as well as numerous affections of the skin, several of which are quite different from those usually encountered by dermatologists, and, I think, can be referred to that group of affections which Brown-

Séquard, Ollivier and Dupuy have described as secondary to hemispheric lesions. Eulenburg has recently shown that trophic disturbances of various kinds may be induced by destroying various cortical areas in animals, and we certainly have a complete parallel to these experiments in the destructive lesions of the cortex in paresis. It is not necessary to refer these peripheral complications to the sympathetic ganglia! Poincaré and Bonnet have endeavored to locate the original lesion of the disease here, but they have not been confirmed. I have found pigmentation of the inferior cervical ganglion in one case, but not in others, and I question whether this change was pathological.

And now as to the disturbance of the delicate associations on which the incoherence and the delusions of grandeur *must* depend.

The cerebral cortex contains, besides the granular elements, whose nervous nature is disputed, pyramidal and spindle-shaped nerve cells. We can partly draw a parallel between the ganglion cells of the retina and those of the cortex. As in the former, a number of individual elements act synchronously as receptive units, when subjected to a luminous impression, so we may suppose the cortical nerve cell to be an impressible organism, and to be associated with its fellows in the reception of appropriate impressions. But there is this difference between the two cases, while the retinal cell is subjected to repeated impressions which successively blot each other, and no permanent registration takes place, we have in the six hundred million ganglion cells of the hemispheres a sufficient number of registering groups to store away all the impressions an individual receives in his life time, and as one group of cells is subservient to one impression alone, the permanency of our recollections is explained. That the registration of impressions can lie in the ganglion cell, we can readily prove by the phenomenon of the retinal after-image. This is a rudimentary remembrance, as it were; it is true that it lasts but a short period, but how unfavorably is the retina organized in this respect as compared with the cortex?

Meynert draws the beautiful parallel that a sensory surface like the retina, receives successive impressions, and transmits

them to the hemispheric cortex for recollection, just as a camera obscura receives successive transitory images, and these are transferred to the different leaves of a sketch-book for permanent preservation by the artist's pencil.

Now let us suppose that we have received an impression by means of the retina, and another by the ear, from the same source, let us suppose the retinal impression to be a cat, the auditory perception, the peculiar cry of the animal. The two impressions are associated, and the shape and voice of the animal constitute a combined conception of the simplest kind. On the next occasion, when we hear a cat without seeing it at all, we have a mental image of its shape, because the auditory perception centre, being in an organic connection with the visual, by an associating fasciculus recalled the latter into action.

We may carry this example further, and substitute more complicated ideas for each of these simpler impressions, then we will be able to make out our case for progressive paresis.

The chief intellectual phenomenon of the paretic (during the quiet interval, in which the real character of the disease can be best studied) is a loss of his proper sense of self-consciousness,*—his sense of self-consciousness depends on his proper appreciation of his surroundings,—the appreciation of his surroundings depends on the association of numerous impressions which have acted on him during his life-time as the teachings of experience and instructors,—and these impressions have become either destroyed, or if they exist, are not associated. To this there is a pathological parallel. At the apex of the convolutions the vascular channels are more free, and the venous, as well as the lymphatic outflows are less apt to suffer obstruction than at the depth of the sulci, for here, as the vessels have to dip deeply between the convolutions, and dense meningeal exudations chiefly occur here, the compressible emunctories suffer. This serves to intensify the atrophic

*This involves an apparent, but only an apparent, contradiction, some might suppose the delusions of grandeur to correspond to an increase of self-consciousness. A minute's reflection will serve to show that a loss of the relations to himself and surroundings, which alone permit the existence of such delusions, are deductions from, not additions to, the paretic's mental life.

and exudative processes in that district whose emunctories are blocked up. Now, it is precisely at the base of the sulci that the associating fasciculi curve around to join neighboring convolutions; this connection being interfered with, the different individual impressions may co-exist, but they are not associated; they are disconnected factors. In short, in the destructive lesions of associating apparatus, we perceive the stamp of incoherence.

It is not always this coarser association that is affected; usually the fifth (eighth in the occipital lobe) layer of gray matter is the first to suffer, and it is here that the sclerotic patches described by Meschede, the miliary sclerosis of Tuke and a curious lesion observed by myself, are located. This layer is made up of spindle cells, which are not connected with the projection fibres, but with the associating band at their poles; it is reasonable to suppose that they represent the aggregate compound of two impressions, and are the seat of ideas abstracted from the impressions on which these ideas were originally built up. We can here perceive why the more intricate mental processes (mental co-ordinations), like the more delicate motor co-ordinations, should become impossible, while the simple registration of impressions, and the recollection of isolated facts, should still be well-nigh perfect. Changes occur in the spindle cells, both in this disease and in chronic mania, much earlier than is usually supposed. Atrophies of their slender bodies cannot be determined very accurately, but on counting the number of these bodies in a paretic's cortex, and comparing the result with the healthy average, they will be found diminished.

It is on the loss of proper self-consciousness that many of the so-called erratic actions of paretics depend; they lose the power of comparison, and consequently create vast projects (never very ingenious), without calculating their means of carrying them out. They commit a hundred actions which might well be characterized as infantile or boyish. Their tendency to lie, for some of their apparent delusions, are nothing but lies with which they finally deceive themselves, is due to that deficient judgment which a loss of associating power always entails.

Beside loss of associating mechanisms, deficiencies of whole recollections take place, with which cortical areas are found destructively involved in their whole thickness. This explains their forgetfulness of certain events, which we have noticed, and also the fact that such oblivion may involve both long past and very recent recollections. In one case, where an area of the cortex was so much destroyed, that the destruction became visible even to the naked eye, as you see in the plate I have caused to be passed around, the association and projection fasciculi were the seat of a molecular degeneration, which I take to be a secondary process, analogous to the changes described by Tuerck. It seems that the association fibres decay when the centres which they serve to connect are destroyed.

Here is a field for speculation which I shall content myself with having touched on, for fear of encroaching on the evening.

One word as to the general results of our observations, as compared with the observations of others, and from which the following deductions can be drawn:

1. Derangements of voluntary motion, and derangements of the intellect, both depend on changes of the cerebral cortex, and they should always be considered together, pathologically and clinically, just as in the combination, for example, of ataxic and amnesic aphasia.

2. Morbid conditions of the cerebral cortex are of two kinds—the first depends merely on acceleration or retardation of biochemical processes in the nerve cell and nerve fibre, dependant on anomalies of the vascular current; as a clinical result we have maniacal excitement or melancholic atonicity It is not yet definitely settled under what concrete conditions we will have one or the other of these manifestations, but from their dependence on essentially evanescent conditions it is readily seen that mania and melancholia are only symptomatic terms. The symptoms of the first stage of progressive paresis belong, in great part, to this group. The second class of morbid conditions depends on processes which are destructive, permanent and irreparable. It is indifferent what the character of the change may be, its results depend on the region affected.

3. It is possible for marked paresis, even to complete paraparesis, to occur without any spinal lesion whatever. A spinal lesion is neither essential to, nor characteristic of the typical disease. Where it does occur in the typical affection, it is a secondary process, not a pronounced lesion, it is an atrophy dependant partly on the long continued disuse of the motor tracts, partly on the increased lateral pressure, exerted by the accumulating cerebro-spinal fluid. We know since Magendie's time that the spinal arachnoid space, being capacious, and surrounded by compressible walls (venous plexus), acts as a safety-valve to encephalic pressure. But with the frequent repetition of the fluxions causing the latter, the safety-valve itself suffers, and with it the nutrition of the cord. Where the spinal lesion is in the posterior columns, we have a complication to deal with, as has been specified. The fact that in the earlier stages of progressive paresis no changes in the electro-muscular contractility, and no anæsthesia occur, proves that the paresis is a true paresis of the will-power, and is not of a spinal nature. An affection of the medulla oblongata and the mesencephalon, may take place quite early. Oculo-motor ataxia, facial and hypoglossal paresis, must occasionally be referred to the central tubular gray matter, not to the cortex.

4. The incoherence in articulation and in the construction of sentences, their stopping in the middle of one sentence to pick up another at the wrong end, must be referred to the most elaborate associating system we possess, namely the *claustrum*.

I have refrained from detailing the lesions affecting these localities, histologically, since I may have to refer to them in connection with the specimens exhibited. I would simply state, that many authors have described as lesions, appearances found in every individual who has passed his twentieth year, and others have registered and even photographed the results of the reaction of absolute alcohol, (leucin and cholestearin precipitates) as lesions characteristic of progressive paresis. My results, with the exceptions to be detailed, are mainly in accordance with those obtained by Rokitansky, Westphal, Meynert, Adler, Lubimoff, Schuele, Magnan, Mierzejewski, and Herbert Major.

a. The so-called colloid bodies are not strictly to be so termed; they are evidently a fusion of several organic compounds, among which lecithin is probably one.

b. Fat or oil globules, I have never found.

c. Amyloid degeneration of the vessels occurs in the luetic form alone, as far as my experience goes.

d. There is no correspondence between the preponderance of melancholic symptoms and the development of protagon spheres. (Schnele.)

e. Pigmentation of the cortical nerve cell, where diffuse, can not be unquestionably interpreted as abnormal, since the cells of the substantia nigra and locus coerulens are always pigmented. But pigment clumps in a nerve cell are decidedly pathological. These do not constantly occur.

f. Miliary aneurisms, fusiform and dissecting, must be shown to have a definite relation to the symptoms of the disease, before they can be considered of any importance. I am convinced that where the investigator has found them very frequently, he has caused their artificial production by pursuing improper methods. I have not found a single clear appearance of this kind which would stand all tests. Besides they occur in those who never have been insane, as a predisposing cause of apoplexy.

g. There is often an apparent disproportion between the gravity of the mental symptoms and the changes in the ganglion cells, which appear to be intact. In such cases it is the rank growth of the connective tissue, which with Rindfleisch, we may consider as having rendered the delicate associating and projecting machinery of thought useless.

With regard to the frequency of the perivascular dilatation, it, with several affections of the tutamina cerebri, is an important etiological factor. We know that the existing causes of progressive paresis are all such as will cause vascular determination and retarded lymph-outflow. The ill-supported vessels can no longer be held in tone by the vascular centre, this finally breaks down by sheer exhaustion. We correspondingly find that, in accordance with the resulting engorgement, the disease begins with a condition of excitement, not the melancholia, which Guislain claimed for it more in accordance with

the formulas which he followed, than with the clinical facts.

We have thus traced clinical groups of symptoms, which are constantly associated with complicated central changes to these as their origin, I have been more or less theoretical in my deductions, it must be recollected that the nature of the subject forbids any other method of treatment.

Although psychological pathology, the highest branch of neurological inquiry, is still in its earliest infancy, and all statements as to its possible future degree of perfection, are therefore premature, we may make one prediction with tolerable confidence: That a palpable proof of the exact manner in which disorder of the higher elements of the nervous system determines aberrations of thought and of action, will forever lie beyond the pale of human demonstrative power!

Still it would be an excessive conservatism, which resting satisfied with this discouraging conviction, would forbid further investigation in our field because it is partly speculative.

By logically combining the results of anatomical, experimental and embryological research, we are enabled to offer an explanation for the phenomena of the mind, which without being comparable to a mathematical solution, is yet something more than a new theory. For an explanation has passed out of the realm of the wholly hypothetical as soon as the contingent probabilities in its favor, become as numerous and powerful as they have become in the case of Meynert and Wundt's propositions.

Finally as an apology, for having approached the vast and problematical subject of the mind, vast and problematical even in its crippled state, I may venture the suggestion, that if it is worth while studying insanity at all, it is worth while studying it in accordance with those branches without which no specialty is a scientific study, namely, Anatomy and Physology.